

PHILADELPHIA NEUROLOGICAL SOCIETY.

May 31st, 1897. The President, Dr. Charles W. Burr, in the chair.

Dr. Charles K. Mills presented a

CASE OF FACIAL SPASM (PAINLESS TIC).

The patient, a man fifty-five years old, was brought to Dr. Mills by Dr. Alexander R. Craig, of Columbia. The spasm was of three years' duration, and had gradually increased in severity during this time. All the muscles on the left side of the face, supplied by the seventh nerve, were affected, and at the height of the attacks blepharospasm was very marked. The paroxysms came on at short intervals, and the patient had several during the short time he was before the Society. He had suffered with what he described as "vertigo," which had caused him to fall six or seven times during the last twenty-five years, and he had suffered also at intervals during this time with headache. He had polyuria, but examination showed neither albumin nor sugar. Examination with the ophthalmoscope revealed no changes in the eyeground. For several weeks he had been treated by increasing doses of coniin hydrobromate. One-fiftieth of a grain three times daily was given at first, and the amount was gradually increased until he took as much as the one-fifth of a grain five times daily. The treatment had no perceptible effect in reducing the spasm, and the only constitutional symptoms produced were unusual weakness in walking and a feeling of general debility.

Dr. Wharton Sinkler exhibited a case of

ERB'S PARALYSIS (UPPER ARM TYPE)

in a child of seven years. He stated that the history of traumatism made the diagnosis between Erb's juvenile form of muscular dystrophy and paralysis of the brachial

plexus, of the upper arm type, easier than it would otherwise have been. The patient, a child of seven years, born in a healthy family, attempted two years ago to emulate a trapeze performance which he had seen in the circus. He was found hanging by the arms from a trapeze, with his head down and his legs turned back over the body in a position which produced great strain upon the upper limbs. About two months later the parents noticed that the boy did not use his right arm as freely as the left, and an examination showed that the shoulder and chest muscles of the right side were much wasted, but that no sensory disturbances were present. Pain had not been complained of at any time. The only electrical alterations noted were in the lower third of the pectoralis major muscle, which did not respond to either current. The atrophy was supposed to be due to an injury of the upper roots of the right brachial plexus.

Dr. James Hendrie Lloyd:—This is strikingly like a case that I have in the Philadelphia Hospital. The patient, a man, has complete absence of the pectoralis major and minor muscles. In his case the defect is apparently congenital. I have had occasion to go over the literature, and I have found that these cases are not so extremely rare. My patient is a large, able-bodied man. He has perfect movement of the arm in every direction except adduction across the chest. He has always done hard work and has experienced no disability from the absence of the muscles.

Dr. H. N. Moyer, of Chicago:—I also have seen a case in which the lower portion of the pectoral muscle was entirely absent, while the clavicular portion was intact. I regarded that case as congenital.

Dr. James Hendrie Lloyd presented

A CASE OF TETANY.

The man, an Italian, had only been in this country a few years, and had had a similar attack once before. The symptoms were so characteristic that the diagnosis could hardly be questioned. The case was one of great interest on account of the rarity of the disease in this country.

Dr. Hugh T. Patrick, of Chicago:—In the well-marked cases of tetany which I have seen, the spasm could be produced by the retardation of the circulation caused by simple

elevation of the arm. This would show that pressure on the vessels, as well as on the nerves, may cause the spasm, that is, the continuous cramp which is the usual manifestation of the disease.

Dr. William G. Spiller:—I have seen, I suppose, at least fifteen cases of tetany. "This is not an unusual disease in Vienna, where it occurs almost endemically in the early spring months. Pressure on the arm, over the nerves and vessels, sufficiently long continued, usually produces the spasm (Trousseau's sign). This is readily demonstrated in the present case, which is one of the most typical that I have seen. It is a disputed point whether the spasm is caused by pressure on the arteries or on the nerves.

Dr. Dercum presented a case of

UNILATERAL ATAXIA AND TREMOR,

the diagnosis being undetermined, and resting between multiple sclerosis and syphilis.

H. A., aged forty, white, a Swede and a sailor, was a patient at the Philadelphia Hospital.

Family History.—He always enjoyed the best of health until about seven years ago. He has followed the sea for the last twenty-five years. Six or seven years ago he had a chancre, and three years later suddenly lost power in the right side of the body. The hemiplegia was slight and disappeared altogether in four weeks, and was not accompanied by loss of the power of speech. The symptoms of the present trouble, consisting of headache, vertigo, drowsiness and diplopia, first showed themselves last December (1895). About that time he suddenly lost power in the left side of the body, but the attack of weakness was not accompanied by loss of consciousness. He recovered power on this side quite rapidly, but ever since has had difficulty in walking and has not been able to hold objects well with his left hand.

Present Condition.—(Nov. 5th, 1896)—The sway is decidedly increased on making the Romberg test. He stands unsteadily when resting only upon the right foot, and is entirely unable to stand alone upon the left, on account of ataxia and not of weakness. It is observed that the left foot is swung awkwardly upward and outward in walking, and that it strikes with the flat of the sole upon

the ground. The movements of the right foot and leg appear to be normal. The patient, lying upon his back, and being instructed to perform certain movements with his legs, shows marked ataxia of the left leg, and a very slight degree of ataxia of the right. The ataxic movements of the left leg are somewhat jerky in character. Movements of precision are well executed with the right hand, but there is a slight unsteadiness of the right upper limb as a whole. The movements of the left arm are excessively ataxic and jerky. When the patient is told to touch the nose with the tip of the left forefinger, the arm is clumsily moved until it approaches the nose, when lateral, and to and fro, oscillatory movements, jerky in character, supervene, and become more intense with the degree of effort made. Tremor is manifested in the left hand upon voluntary motion, and consists of coarse up and down movements.

The face bears a fixed expression of somnolence; the eyebrows are slightly raised, giving the patient the appearance as though he were making an effort to keep awake. The lines of the face, with the exception of those of the forehead, are comparatively smooth, and the features appear somewhat relaxed, as in paresis. The head is held slightly inclined to the right shoulder. No signs of facial palsy or twitching, and no tremor of the lips are noted. The tongue is protruded slowly and its movements are slightly irregular. The pupils respond normally to tests, and are equal in size. The movements of the eyeballs appear to be as extensive as normal. Marked vertical nystagmus can readily be elicited. The right eyelid seems to droop slightly, and the right palpebral fissure is smaller than the left. Neither anesthesia nor affection of the sphincters is noted.

Speech is somewhat scanning and drawling and reminds one of that of multiple sclerosis. The knee jerk is plus on the right side and normal on the left. There is slight ankle clonus on the right side, but none on the left. The elbow jerk, biceps jerk and wrist jerk on the right side are plus; on the left side they are normal.

The plantar reflexes are marked, the left excessively so. There is no toe reflex. The superficial reflexes of the thigh and abdomen are much increased. The patient had headache, dizziness and ringing in the ears for five years, until the fall of 1895, when he was relieved by medicine, and has

no dizziness now. He has no attacks of any kind, and no pain in the bones. His mental condition is fair. He can give a good account of his past life and of his present symptoms; he is neither expansive nor depressed at any time, has no headache, and has never had any delusion.

Dr. Dercum thought that the case looked at first sight very much like one of multiple sclerosis with unilateral symptoms. In favor of this view were the coarse intention tremor and the scanning speech. If this view is correct, it would, perhaps, point to a possible relation between multiple sclerosis and syphilis. The syndrome, however, is certainly not that of syphilis, and whatever the character of the lesion may be, it is not in all probability that of ordinary nervous syphilis. The question of paresis was also taken up and briefly considered, but this diagnosis was considered improbable.

Dr. Wharton Sinkler:—This case recalls a patient who was under my care some years ago, and suffered from disseminated sclerosis with marked intention tremor on both sides. He had a cerebral hemorrhage with hemiplegia on the left side, which developed while he was under treatment in the Philadelphia Hospital for the multiple sclerosis. He partially recovered from the paralysis and the tremor disappeared entirely on the hemiplegic side, while it remained unchanged on the other. May there not be a distribution of the sclerotic patches in areas which inhibit the intention tremor on the one side in Dr. Dercum's case?

Dr. Henry S. Upson, of Cleveland:—It is a well-known fact that lesions in the great basal ganglia may cause intention tremor. Lesions about the optic thalami may cause symptoms similar to those of paralysis agitans. This would suggest that, perhaps, in this case there is a lesion near or in the great basal ganglia which causes these symptoms. It may be impossible to make a diagnosis between such a lesion as I have spoken of and the sclerotic patches of ordinary insular sclerosis.

Dr. F. X. Dercum presented a case of

RIGHT HOMONYMOUS HEMIANOPSIA

without visual or optic aphasia, following an apoplectic attack. The history of this case is briefly as follows:

H. H., a patient at the Jefferson Hospital, aged forty-five, was seized with dizziness and vomiting two years ago while at work in his garden. He soon became unconscious

and remained so from ten o'clock in the morning until three in the afternoon. On recovering consciousness he found himself in bed. He was able to speak, but had some weakness of the right side. A curious optic illusion was, however, present; he felt as though the ceiling were much lower, and were arched over his bed. This illusion persisted for several days. A subjective numbness of the left leg below the knee was also noticed.

At present the patient presents a typical right homonymous hemianopsia with preservation of central vision, but no hemiplegia, and no anesthesia. He states that the sense of numbness, which made its appearance in the left leg in the beginning of the attack, has steadily persisted. It does not appear to be pronounced, and cannot, of course, be verified by any physical examination. The heart and vessels are normal. The case is interesting as an instance of an apoplexy, in all probability resulting from a cerebral hemorrhage, in which every symptom, save the hemianopsia, has disappeared; and also because of the absence of visual or optic aphasia.

Dr. Quimby, of New Jersey:—It seems to me that the sudden attack would point very strongly to a diagnosis of hemorrhage. I have seen a similar case in which the necropsy showed a cortical hemorrhage.

Dr. Charles W. Burr presented

A CASE OF HEMIPLEGIA (POSSIBLY HYSTERIC) WITH
ANKLE CLONUS.

The patient was a single man about twenty-seven years old and a laborer. He denied having had syphilis and having indulged in alcoholic excess. In September, 1892, while at work he suddenly became dizzy, fell down unconscious and remained in this condition for six weeks. On return of consciousness he was found to be speechless and paralyzed on the left side. His jaws were so stiff that he had to be fed with a spoon. After three months speech returned, and at the end of a year he could walk. Since the beginning of his illness he has had several attacks of unconsciousness. At the time of his admission to the Philadelphia Hospital, in 1895, he appeared to be a case of ordinary hemiplegia, and was sent to the out-wards. Some

months later he had an attack in which he screamed, threw himself on the ground, writhed, jerked and bit, but did not become unconscious. He was sent to Dr. Burr's ward and became quiet in a few minutes after his arrival. For some days he lay apparently perfectly conscious and seeming to hear and understand all that was said to him, but unable or unwilling to move or speak. On the eighth day he became very religious in his actions, clasped his hands and looked heavenward or held an open prayer-book hour after hour without turning the leaves. The next day he was sentimental and wished to kiss the hands of those about him, and finally was somewhat maudlin. Speech then returned, and at first he spoke in whispers, but in a few hours he talked well and got up and walked about the ward as usual.

Examination showed that he was a strong, well-built man. The heart, kidneys and, indeed, all the thoracic and abdominal organs, were normal. Very marked spastic hemiplegia of the left side was noted. The left knee-jerk was exaggerated. There were regular and persistent wrist, patellar and ankle clonus, but no muscular wasting. The sensory symptoms were remarkable; at times there was complete left hemianesthesia, stopping abruptly at the median line and involving the mucous membranes; at times this anesthesia disappeared completely, and at other times affected only a part of the leg or arm, the distribution then having no regard to nerve supply, but resembling a glove or stocking. The same was true of the pain sense, which, however, was never completely absent. At first the eye examination was negative, but later Dr. Oliver discovered contraction of the fields, involving especially the left side, more marked in the left eye, and affecting chiefly the left upper quadrant. The paralytic symptoms have remained constant, but, as stated, the sensory have varied greatly.

Dr. Burr thought the man presented many of the symptoms of hemiplegia, but that this diagnosis did not explain all his symptoms. The fit certainly was hysteroidal, and he knew of no organic disease which would account for the strange variation in the anesthesia. He believed, therefore, that the man had hysteria, but was by no means sure that it was not added to an old organic hemiplegia. Until recently he would have said that the great increase in the deep reflexes, and especially the existence

of true ankle clonus, were absolute proof of the organic origin of the hemiplegia, but he was beginning to be more than doubtful of the truth of that view, and had presented this man to obtain an opinion on the question.

Dr. Charles K. Mills:—I do not believe that this is a case of hysteria pure and simple. I believe that the man is suffering from an organic disease with possibly hysteria superadded. I would say in regard to his vacillating symptoms, that I have seen variations in the sensory phenomena in cases of syringomyelia similar to those presented by this man.

Dr. Hughes, of St. Louis:—Some years ago I was interested in a medico-legal case in which Dr. Moyer was also concerned. In this case, the question of ankle clonus was an important element in the diagnosis. I have taken the position that the profession has been largely at fault on this matter of hysteria. The general ruling in diagnosis is either all hysteria or no hysteria, yet it is only reasonable to expect, and if we observe our cases closely we will find it true, that the gravest kind of lesions bring out latent hysterical manifestations. I think that we have done ourselves and our patients great injustice in assuming that hysteria may not coexist with grave organic disease. Gowers has taken the same position. I believe that if in this case the history is carefully inquired into there will be found a hysterical tendency in the family. I do not doubt the existence of organic lesion in this case.

Dr. H. N. Moyer, of Chicago:—The case referred to by Dr. Hughes was a very interesting one. The question was whether the trouble was functional or organic. I leaned to the view that it was organic. The patient had ankle clonus. The man's present condition after a period of ten years confirms the diagnosis of organic lesion. He is in the condition of a person after a moderately severe attack of transverse myelitis.

Dr. Hugh T. Patrick, of Chicago:—The question of clonus interests me greatly. I have examined a number of cases of hysterical clonus. The clonus in this case seems to be that of hysteria. It started at the rate of about three hundred and sixty movements a minute, which is the ordinary rate of organic clonus, but before the termination of a third of a minute it had fallen to a hundred and twenty a minute. As far as my experience goes this change in the rate is characteristic of hysterical clonus.

Dr. J. H. W. Rhein:—Apropos of the question of ankle clonus in hysteria, I should like to present again the case reported by me at the last meeting of this Society. The history is briefly as follows: The patient, after inhaling benzine vapor while cleaning a manure tank, became partially unconscious, and soon afterwards had tremor in the entire body. A month

later, when first seen by me, he had spasticity in the legs, general tremor when walking, spasmodic strabismus, segmental anesthesia, and a true ankle clonus, which persisted on one side, but was easily exhausted on the other. Under hypnotism the ankle clonus disappeared. Dr. McCarthy, who has hypnotized the patient frequently, saw it disappear under suggestion. It is not now present. As the patient walks you will notice that he has lost the spastic gait and the general tremor. All the symptoms, including the segmental anesthesia and the spasmodic strabismus, have disappeared. The diagnosis is clearly hysteria and the case demonstrates beyond a doubt that in this affection we may have a true ankle clonus.

Dr. A. A. Eshner:—I should like to ask a question which suggests its own answer. If hysteria may develop entirely apart from organic disease, is it less likely that it should occur in connection with organic disease, and need the presence of organic disease exclude the existence of hysteria, or the presence of hysteria exclude the existence of organic disease? My view of Dr. Burr's case is that it is organic, whether primarily or secondarily, I cannot say. Whether, in addition, there is hysteria, it is difficult to determine, but I am inclined to think that there is.

Dr. Martin W. Barr, by invitation, exhibited

AN EXTRAORDINARY CASE OF ECHOLALIA.

Dr. Charles K. Mills:—Had we time this paper would open up a wide field for discussion. It seems to me that Dr. Barr, like myself, has perhaps described two affections which are radically different, although I do not know that he intended to include the case exhibited in the first class referred to by him. The cases of echolalia and of ejaculatory speech of various kinds, where the attacks come in paroxysms, are more of the nature of obsessions, and are not cases of true echolalia, as this seems to be.

One other point. Probably no one has taken more interest than I have in the attempt to localize special parts of the brain related to the function of speech, yet in a case of this kind too much stress may be laid upon the effort to definitely fix the position of the lesion to explain the symptoms. The case would seem to me to be better explained by the supposition of arrest of development of large areas of the brain, although, of course, even in this condition, there may be a preponderance of the arrest, or of the lesion, in certain parts.

Dr. Charles K. Mills exhibited

A CASE OF PARALYSIS OF THE ELEVATORS OF THE
EYEBALLS.

J. F. B., aged thirty-seven, white, single, and residing in West Chester, Pa., was sent by Dr. Ellwood Patrick to Dr. Mills for consultation.

The patient's ocular disturbance began four years ago, and vision had been getting weak. He had had diplopia during the past year, one image being situated a little above the other. There had been no history of pain. Since last fall there had been disturbance of locomotion, and the patient had had difficulty in raising his feet from the ground. Visual symptoms had been noted for two years. The man could not raise his eyeballs above the horizontal plane in attempting upward rotation, and in doing this one or the other eyeball tended to move slightly outward. Lateral and downward movements appeared to be good. The movements of the upper eyelids seemed to be well preserved, but the inability to move the balls upward gave the appearance of partial ptosis. The patient complained of a general feeling of weakness in the legs. He had not had lancinating pains, and his knee-jerks were exaggerated, and although there was no persistent ankle clonus, a slight spurious one was noted on the left side.

Dr. Hansell reported that there was decided concentric limitation of the right field, and positive, but slight, concentric limitation of the left. The paralysis of the upward rotators was complete. There was divergence of the left eyeball, but no lateral paralysis. The pupils were equal and reacted sluggishly to light and in accommodation individually and consensually. The media and fundus in each eye were healthy. The central visual acuity was 20/40 R. 20/30 L. Diplopia, with the second image a little above the first, and to one side, was noted when the objects were in certain positions. The gait was peculiar, in that the patient did not follow a straight line, which was possibly due to false projection. There was slight, almost imperceptible, difficulty in speaking. The tongue was protruded somewhat to the left, and there was some tendency to drooling.

Dr. Joseph Sailer reported

A CASE OF GLIOSIS CEREBRI.

Dr. Wm. G. Spiller read a communication in the name of Dr. G. Marinesco, of Paris, on

THE PATHOLOGY OF MORVAN'S DISEASE.

The case of Morvan's disease, which I have reported to the Société des Hôpitaux, was a well-defined form of the disease, and affords, in my opinion, a solution of the pathology of this affection. The clinical diagnosis was made by Charcot. The pathological examination, made by me, has only revealed a lesion of the gray matter, involving especially the posterior horn, and, to a slight degree, the anterior also, at least in the lower cervical region. In the portion of the cord affected in this case is the centre for the sensory, motor and vasomotor innervation of the hand. The nerves are intact. I have been led to conclude, therefore, that the trophic disturbances of Morvan's disease in my case should be referred to the most important lesion, viz.: the alteration of the posterior horn. Furthermore, in this case the lesion was unilateral and on the same side as the trophic disturbances, the nerves were intact, and the lepra bacillus was not found. How can these conditions be otherwise interpreted? A question might only arise as regards the alteration of the anterior horn. The condition here may possibly have had some effect on the symptoms, but the changes in this horn were not very important, and their location does not explain the trophic disturbances. One is forced, therefore, to admit that the cause of these trophic lesions is to be found in the condition of the posterior horn or, more exactly, of the intermediate and posterior gray matter. In the light of our present knowledge it seems to be an established fact that the alteration of these regions may cause cutaneous and osseous trophic disturbances. These, however, are produced reflexly, as I have shown several times in my papers. Leyden and Goldscheider, and Brissaud have adopted my views.

Although I have determined that Morvan's disease is due to destruction of the posterior gray matter, it does not

follow that other lesions cannot cause an analogous clinical picture. The explanation which I offer in regard to the nature of Morvan's disease in my case seems to me the only possible one. At all events, the symptoms cannot be attributed to an abnormal condition of the peripheral nerves, as these were intact.

Dr. Spiller:—Dr. Dercum and I have been unable to believe that in every case in which the posterior horn is destroyed in the lower cervical and upper thoracic regions the symptoms of Morvan's disease are present. We reported, in the *American Journal of the Medical Sciences* (December, 1896) a case of syringomyelia in which the posterior horn of one side was destroyed throughout the cervical region, and also in the upper thoracic region, but no symptoms of Morvan's disease had at any time been detected. Dr. Marinesco has made so many valuable contributions to neurological literature that we could not lightly pass over an apparent discrepancy in our findings and the interpretation of them. We have, therefore, requested an explanation and he has kindly sent the above statement.

We cannot judge from our case of the nature of the lesions produced by destruction of the intermediate gray matter, that is, of the substance between the anterior and posterior horns, as the cavity does not extend far into this region. It is true, however, that most of the fibres passing into this portion of the cord from the posterior roots are destroyed, but if trophic cells are situated here their axis cylinders may pass to the periphery of the body by way of the anterior roots. This may possibly explain the differences in Dr. Marinesco's case and ours.

In another communication Dr. Marinesco has called our attention to the fact that in our case an arthropathy of the shoulder existed on the same side as the cavity in the posterior horn, and, if we understand his meaning correctly, he attributes the disease of the shoulder joint to the destruction of the posterior horn. This may possibly be the correct interpretation of our case, but we prefer to be somewhat cautious in the full acceptance of this explanation until other cases are reported.